JACC Vol. 22, No. 2
August 1993:632–3

LETTERS TO THE EDITOR

Sestamibi in Myocardial Perfusion Imaging

Melon et al. (1) are to be congratulated for helping to demystify the relative merits of technetium-99m sestamibi and thallium-201 for myocardial perfusion imaging. I wonder whether an additional insight can be drawn from their data. Although I agree that "the decrease in b with time of measurement . . . is consistent with increasing underestimation of tracer extraction (through PS) as the fraction of tracer undergoing back diffusion increases" is an appropriate observation in relation to thallium, I do not believe that this is necessarily true of sestamibi. In fact, examining the data presented in Figure 2A, and consistent with the clinical observation that sestamibi washout from the myocardium is slow, it would seem that the time-related fall in b with respect to sestamibi might be under a different influence.

After 20 min of flow >3 ml/min per g the slope of sestamibi retention versus flow does not differ from zero. At the same time, unlike thallium retention, sestamibi retention at high flow does not appear to decrease. Rather, retention at low flow levels appears to increase, thereby reducing the slope of the retention flow relation. I believe that this is consistent with ongoing extraction of sestamibi preferentially in low flow segments (where the myocardial to blood pool tracer concentration gradient is lower). In this light, I wonder if the authors would agree with the conclusion that the decrease in b might be consistent with two distinct phenomena: 1) increasing underestimation of thallium extraction as back-diffusion increases, and 2) relatively increased net retention of sestamibi in relation to lower flow.

I believe that this distinction has clinical importance. The observation that sestamibi retention becomes disassociated from flow in this way suggests unique potential for its use in assessment of myocardial viability in the presence of critical hypoperfusion ("hibernating myocardium").

R. J. BURNS, MD, FACC
Division of Cardiology
The Toronto Hospital
399 Bathurst Street
EW5-552
Toronto, Ontario M5T 2S8, Canada

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Reply

In our study, tracer retention in the myocardium was quantified by relating tissue tracer concentrations at various time points after injection to the arterial input function. The calculated tracer retention index reflects the combined effect of initial tracer uptake and subsequent washout from myocardium. Early after tracer injection, this retention index is more closely related to delivery of the radiotracer; later time points represent equilibration of activity between vascular and tissue space. Because technetium (Tc)-99m sestamibi is retained in tissue with a much longer biologic half-time than that of thallium-201, it is possible that tracer uptake continues in low flow areas, resulting in net accumulation and, hence, overestimation of flow. However, this continuing uptake of Tc-99m sestamibi depends on a concentration gradient of the tracer between vascular and tissue space, which may exist only for a relatively short time period after injection. Recent observations of Sinusas et al. (1) of Tc-99m sestamibi redistribution in an animal model of sustained low flow ischemia support this hypothesis.

Burns' notion about the possible clinical role of Tc-99m sestamibi for the assessment of tissue viability is currently the subject of several investigations. However, first reports (2,3) comparing Tc-99m sestamibi with either thallium-201 reinjection or positron emission tomography–F-18 deoxyglucose imaging suggest limited diagnostic accuracy of Tc-99m sestamibi in the assessment of tissue viability in patients with advanced coronary artery disease. At this point, further studies are required to define the best Tc-99m sestamibi imaging protocol to enhance its role as a marker of viable myocardium.

PIERRE G. MELON, MD
TIMOTHY R. DEGRADO, PHD
NEIL A. PETRY, RPH, MS, BCNP
ROB S. BEANLANDS, MD
NGOC NGUYEN, BS
MARKUS SCHWAIGER, MD, FACC
Division of Nuclear Medicine
University of Michigan Medical Center
1500 East Medical Center Drive
Ann Arbor, Michigan 48109-0028

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Torsades de Pointes

In his very pertinent comments on the report by Buxton et al. (1) on polymorphic ventricular tachycardia, El-Sherif (2) helpfully clarifies the difference between this disorder and torsades de pointes by stressing the importance of the long QTU syndrome in the latter. I believe, however, he should also have stressed the importance of intense bradycardia, associated with atrioventricular (AV) block in most cases, as described originally by Dessertenne (3) and as stressed by ourselves (4,5) in the first English language descriptions of this entity. Indeed, as Fontaine (6) has recently reminded us, Dessertenne's first patients (3) were referred for pacemaker implantation because of complete AV block or major bradycardias. This point does, of course, have therapeutic as well as physiologic

0735-1097/93/$6.00

significance and its mention is intended only to enhance the helpful clarification by El-Sherif (2).

It is interesting to note that Fontaine and his colleagues (6) have been able to confirm that under certain circumstances torsades de pointes could be induced by the ventricular extrastimulus technique (7), though we do appreciate that this is an uncommon situation (8).

JACC readers intrigued by the uncertainty of rendition of torsade(s) de pointe(s) will see that there are different opinions on the need for an "s" at the end of each word, according to Robert Slama and Paul Puech of Paris, as reported by Fontaine (6). The former thinks that the "s" is optional with torsades but that the pointes is invariable, whereas the latter thinks on converse lines believing that we are talking of the twisting(s) of a single spike. Vive la différence!

DENNIS M. KRIKLER, MD, FACC
Cardiovascular Division
Royal Postgraduate Medical School
150 Du Cane Road
London W12 ONN, England, United Kingdom

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Right Bundle Branch Block, Persistent ST Segment Elevation and Sudden Cardiac Death

The report of Brugada and Brugada (1) adds eight more cases to a clinical and electrocardiographic (ECG) syndome previously described in detail by our group (2,3). Patients 1, 3 and 4 in our report (2), affected by aborted sudden death and a mild form of right ventricular cardiomyopathy (4), had the discussed ECG pattern. We demonstrated by electrophysiologic mapping that this ECG pattern is related to late depolarization of the outflow tract of the right ventricle (3). This observation was confirmed by a positive late potentials study on signal-averaged electrocardiography (5) and could constitute the basis for reentry.

Other similarities between the studies derive from the clinical history, as two of our patients and four in the series of Brugada and Brugada had some familial involvement.

The difference between the two reports is substantial, however, as we have described in detail structural cardiac abnormalities in this syndrome, whereas Brugada and Brugada believe that the disorder is functional. However, biopsy was performed in only half of their patients, autopsy was not performed in the patient who died and no quantitative details regarding echocardiography and angiography are given.

We believe that their report confirms that the problem of "cryptogenetic" or idiopathic ventricular arrhythmias (6) is still far from resolution, as there is no standard criterion to define what is normal. We hope that a closer international cooperation will be instituted to examine all these cases, propose an investigational protocol and define diagnostic criteria.

BORTOLO MARTINI, MD
ANDREA NAVA, MD
BRUNO CANCIANI, MD
GAETANO THIENE, MD
Department of Cardiology and Pathology
University of Padua
Via Giustiniani
35100 Padua, Italy

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